Multidisciplinary team aids understanding of Hepatitis C virus and possible cure

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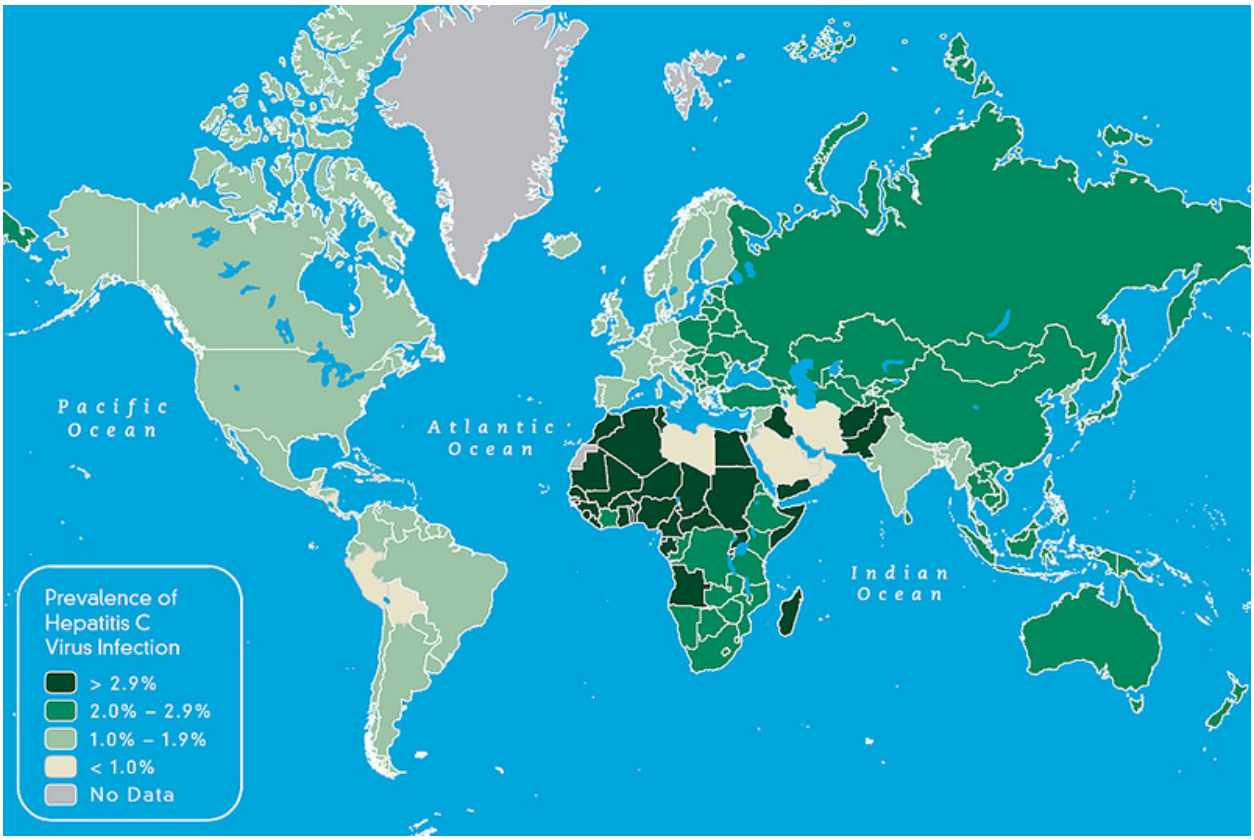

Lab researchers are teaming with others across the country and around the world to understand how a potent new drug works and provide a better understanding of how the virus reproduces in an effort to better target future treatments.

The virus under scrutiny is the hepatitis C virus (HCV) that affects about 150 million people and is the leading cause of cirrhosis, liver cancer and liver transplants. It causes roughly 350,000 deaths each year.

A drug developed by Bristol-Myers Squibb appeared to damage the virus—within 12 hours of treatment—but the mechanism by which it accomplished this was not well understood. Treatments prior to 2011 had modest results and fewer than half of treated patients were fully cured of HCV. A better understanding of how this specific antiviral worked could lead to the development of more effective drugs.

“Unraveling how this drug could cause such a rapid drop in the amount of virus in an infected person’s blood could greatly enhance the ability to design optimal drug therapies and ultimately cure this disease,” said Alan Perelson, with the Lab’s Theoretical Biology and Biophysics group.

A mathematical technique called “viral kinetic modeling” seeks to characterize the main mechanisms that govern the virus’ response to treatment. These computer simulations showed the drug in question blocked two distinct processes (like other antivirals), but also the release of the virus from infected cells. The research results indicate that daily viral production could be four times larger than previously thought which has implications for the development of mutations that could lead to drug resistance.

Other researchers on the team include Susan L. Uprichard and Natasha Sansone from University of Illinois at Chicago; Harel Dahari, Thomas Layden and Scott J. Cotler from Loyola University, Chicago; Richard Nettles from Bristol-Myers Squibb and Jeremie Guedj from Institut National de la Santé et de la Recherche Médicale, France.

The National Institutes of Health, National Science Foundation and University of Illinois Walter Payton Liver Center Guild funded the research.

To view a technical paper on the subject, go to “Modeling shows that the NS5A inhibitor daclatasvir has two modes of action and yields a shorter estimate of the hepatitis C virus half-life.”